TRANSLATION NO. 1262

DATE: 4 Jan 1964

DDC AVAILABILITY NOTICE

~~Qualified requestors may obtain copies of~~ this document from DDC.

This publication has been translated from the open literature and is available to the general public. Non-DOD agencies may purchase this publication from the Clearinghouse for Federal Scientific and Technical Information, U. S. Department of Commerce, Springfield, Va.

DEPARTMENT OF THE ARMY
Fort Detrick
Frederick, Maryland

TRANSMISSION OF VIRUS DISEASES BY SEED

Postepy Nauk Rolniczych (Advances in the Agricultural Sciences) No 2, Vol 86, 1964, pages 41-56

Vladyslaw Blaszczak

Virus diseases of plants are disseminated among plants by different methods. Among these the most important role is played by insects, called vectors in virology. We now have considerable knowledge of the facts in this area. The insects which transmit viruses belong the various orders. For example, one might mention thrips, coleoptera, hemiptera, orthoptera, aphids, and others. However, the most important are aphids and orthoptera. Klinkowski (1948) reports that aphids transmit 37 viruses, orthoptera 32, and other insects 16. However, the role of aphids is considerably greater than one would think from the figures given because orthoptera are characterized by great specialization and transmit only particular viruses and at times on their own particular variety. On the other hand aphids do not show this specialization (poly-valent vectors) and transmit many viruses. For example Doralis fabae transmits around 25 viruses, D. frangulae around 30 and Myzodes persicae more than 50.

Another important means of dissemination of virus diseases within plants is mechanical transportation. This is a matter of abrasion of plants and minor injury caused by different farming operations. We have in mind such operations as harrowing and weeding, cultivation of potatoes, removal of side shoots from tomatoes, etc. In this manner harmful viruses of potatoes, tomatoes and other plants are disseminated. This has given rise to a tendency to limit cultivating practices, for example, in the raising of potatoes.

And yet how are plant viruses transmitted from year to year? As disease causing organisms they do have the ability to produce dormant stages such as fungi in order to survive through the winter or drouths in other climates. Viruses remain so to speak "bound" to their hosts, and at times to their transmitters and in this manner they are assured of continuity of existence. Virus diseases are transmitted by the devices of vegetable

reproduction, as, for example, tubers, bulbs, shoots, and in the case of biennials by rootstocks. This is one of the most prevalent sources of virus plant diseases and particularly for such economically important species as potatoes, sugar beets, and also strawberries and many kinds of ornamental plants. Another important reservoir of plant viruses are perennials, as, for example, fruit trees (peaches, apples, plums), and winter hardy plants such as melolitus, trefoil, and alfalfa. Other viruses winter in their vectors. This is the case, for example, of aphids which inhabit greenhouses, storerooms, and cellars or insects which spend the winter in the open fields. An an example of this we cite the hemiptera Piesma quadrata which spends the winter as an adult and transmits the virus which causes beet curl. There are other viruses, for example, the virus which causes the dwarfing of rice or the virus which causes clubbing of trefoil leaves which are transmitted by their hosts from generation to generation in their eggs. A certain group of viruses is maintained in the soil, probably in their vectors, nematodes. The tobacco mosaic virus has a curious development. It can survive in dry vegetable matter and even in processed tobacco products for many years and still be able to serve as a potential source of disease. Finally, seed of the host plant can play an important part in the transmission of plant viruses from year to year. This is a problem of great interest from the point of view of biology and of importance for agricultural production. At the present time virology has considerable information on this subject at its disposal, but there still remain many problems which are yet unclarified and whose solutions are still based on hypotheses or speculation.

Transmission of the More Important Viruses by Seed

1. Typical bean mosaic virus. Reddick and Stewart in 1919 reported that this virus is transmitted by seed. They stated that the percent of virus affected seed in individual plants was very uniform and even reached 71%. Later investigations made by many authors established that there were wide variations in virus infection in the seed of individual plants and the average degree of virus infection was usually within the figures of a dozen or so to several score percent (Merkel, 1929; Nelson and Down, 1933; Medina and Grogan, 1961; Bojnansky, 1963).

2. Bean-yellow mosaic virus, although related to the virus of the typical bean mosaic, is not transmitted through bean seed. However, a related form, which causes narrow-leaf in yellow lupine is transmitted by seed, which under our conditions is the most serious source of the disease. Corbett (1958), Ksiazek (1962), Blaszczak (1963) have shown that on an average around 6% of the seed of yellow lupine is infected with virus but according to other authors (Merkel, 1929; Mastenbroek, 1942; Zschau, 1962) the percentage is somewhat lower. The extent of infected seed in particular plants in these cases is also highly variable. Zschau (1962) observed variation in virus infected seed from different samples of from 1.7% to 15.3%. In our investigations based on relatively large samples it was shown that the amount of infected seed which transmits the disease from year to year varied in individual plants from 0 to several score percent. More than 50% of the plants of yellow lupine infected with narrow-leaf did not produce infected seeds (Blaszczak, 1963).

3. Tobacco ringspot virus also infects soybean and is transmitted by its seed. The soybean "Harosoy" infected by this disease in field conditions gave 10% infected seed. Another year seed was collected from 47 infected plants and sowed in a greenhouse. It turned out that the rate of infection for 28 plants was 100%, for seven plants 90-99%, ten plants 80-90%, and two plants 60-69%. The average rate of infected seed was 93%, thus being very high. This is an unusual case of heavily infected seed (Athow and Bancroft, 1959). On the other hand in petunia seed the tobacco ringspot virus infection was recorded at 19.8% (Henderson, 1931).

4. Lettuce mosaic virus. Grogan and Barden (1950) showed that the rate of transmission of the virus by different varieties grown under identical conditions varied from 1% to 8%, while some varieties always transmitted the virus at a higher rate than others. Similar variations in Vigna Sinensis were also observed by McLean (1941). Couch (1955) investigated virus infected seed in four plants of the variety Bibb. The percent of virus infected seed was 4.0%, 7.0%, 10.0%, and 11.3%. The rate of transmission of the virus by seeds of individual plants of the two following generations was very similar. A tabulation of the rate of infection based on around 38,000 lettuce seedlings showed that the average rate of infected seeds and the transmission of the disease by them was at 7.88%. This was therefore similar to the highest rate of infection established by Grogan. On the other hand the variety of lettuce Chestnut Early Giant did not transmit the virus by seed in spite of being subjected to infection.

5. Cucumber mosaic virus. It has been asserted that this virus is transmitted by the seed of the wild cucumber (Micrampelis lobata) and not by the seed of the domesticated cucumber (Smith, 1957). However, this is not completely certain. Also the pumpkin mosaic virus is transmitted by the seed ot M. lobata (Doolittle and Gilbert), 1919). The cucumber mosaic virus is in addition transmitted by the seed of other species of plants. Among others it is transmitted by melon seed at a rate of 8% to 27% (Smith, 1957), and Zschau (1960) has shown that it is transmitted by the seed of yellow lupine at a rate of 21%.

It should be added that in addition to the examples mentioned here of viruses transmitted by seed there are a number of other virus diseases, often very harmful, which are also transmitted in this way. Among these are the barley false spot virus (infected seed from 0 to 58%), tomato mosaic, squash mosaic (Middleton, 1944), peach ringspot, dodder virus (Bennett, 1944), pumpkin virus mosaic (Kendrick, 1934), acute pea mosaic virus (Pozdena and others, 1955) and many others (Bretz, 1950; Cation, 1949, 1952; Cochran, 1950). Crowley suggested that among the several hundred known plant virus diseases there are barely 45 known to be transmitted through the seeds. Furthermore he asserts, basing his position on the literature and his own experiments, that the transmission of viruses in the seeds of papilionaceous plants is not at all more common than in other types which is still believed to be true at the present time.

The Influence of the Time of Infection of the Plant on the Virus Infection of Seeds

The time of the infection of the plant has a significant influence on the virus infection of the seed. Usually the earlier the plant is subject to infection the greater will be the infection of the seed. This has been confirmed in many experiments. Athow and Bancroft (1959) sowed soybean seed collected from plants which at different ages showed infection from tobacco ringspot mosaic virus. They determined on this basis the percent of plants with infected seed and the percent of virus infected seed.

TABLE 1

Influence of the period of infection of the soybean "Harowsoy" by the tobacco ringspot virus on the infection of seed

Age of the plants in days	Percent of plants with virus infected seed	Percent of infected seed
38	78	91
46	54	15
46 - 64*	13	10

*Inception of florescence -- around 56 days.

One can see that a delay in the appearance of disease of only eight days brought a six-fold decrease in the infection of seed. Couch (1955) investigated this phenomenon in lettuce. He innoculated lettuce plants with the mosaic virus at various periods of their development and determined the percent of infected seed. Plants grown from infected seed (Secondary infection) gave seed which was 7.4% carriers of the virus, four week old innoculated seedlings when harvested gave 7.9% infested seed, nine-week old seedlings 4.9%, 13 week 4.6%, and 21 week old plants innoculated during florescence and the formation of seed produced no infected seed at all. The author (1963) studied the influence of the time of infection of yellow lupine by narrow-leaf (of the group of the bean yellow mosaic virus) on the transmission of the disease by seed. The period of the appearance of the disease in plants is shown and then we indicate the transmission of the disease by seed sown in greenhouses.

TABLE 2

Transmission of narrow-leaf of yellow lupine by seed in relation to the period of infection of the plant. (Poznan, 1960)

Stage of the development of the plant at the appearance of the disease	Percent of virus infected seed in the harvest
Inflorescent length up to two centimeters	12.9
Full florescence	11.5
Formation of the last pods on the main stem, flowering on the lateral stems	4.9
Well-formed pods on the main stem and formation of pods on the lateral stems	3.3
Situation as above -- plant apparently healthy -- without evidence of disease	1.1

Here the same regularity is apparent. Early infection of the plants conditions a greater infection of the seed. However, another unfavorable phenomenon is apparent here. Namely, that plants advanced in age having completely formed pods on the main stem and not showing any evidence of disease produced more than 1% diseased seed. From the point of view of epidemiology and disease control this is a very dangerous fact. Such results were obtained also by Zschau (1962), and Zawadzki and Grzybczak (1962). Beans infected in the earliest stage of development by the virus of the typical mosaic also produce more infected seed (Fajardo, 1930). The generalization which is sometimes heard that the infection of plants during florescence does not usually cause virus infection in their seeds (Bojnansky, 1963) does apply to many types of plants which transmit viruses by seed, but it is not a law applicable to all varieties of plants.

The weather during the growth period which influences the growth and development of the plant also influences the infection of seeds. A wet and cold summer which lengthens the growth period of the plant encourages a greater infection in the seeds. Such an unfavorable growth period occurred in the Poznan wojewodztwo in 1961. The average rate of infected seed in yellow lupine which was examined was then 9.6% and in the previous year with normal weather conditions the rate was only 4.1%. It has been asserted that the decisive influence on the infection of seed is often the temperature. Singh and his co-workers (1930) showed that four varieties of barley moderately susceptible to stripe mosaic produced infected seed at a temperature of 20 and 24 degrees centigrade. However, at 16 degrees only one variety gave 3% infected seed while at the former temperature (20 and 24 degrees centigrade) it produced 15% and 24% infected seed.

Development of the Seed and Virus Infection

Pathological changes caused by viruses and observable in infected plants are also often apparent in the seed. Usually there is a decrease in the weight per 1000 seeds and at times there is also a decrease in the germination capability of the seed. It is not always true that a weaker development of seeds must be associated with virus infection and therefore with the transmission of a virus by the seeds. Grogan and Bardin (1950) for example did not assert that there was any dependence between the development of lettuce seed and its infection by a virus. On the other hand McKinney (1951) came to the conclusion that barley seed infected with stripe mosaic virus is smaller, but at the least there is such a tendency in such cases. Middleton (1944) showed that weakly formed, light and misformed squash seed carried mosaic virus at a rate of 0.96% while robust and well-formed seed had a rate of only 0.14% infection. This relationship is even more striking in the case of yellow lupine infected with narrow-leaf. Plants which are infected at an early stage produce few seeds and therefore part of them reach an exceptionally large size. Plants which are infected at a later stage have apparently no decrease in the number of seeds but they are lighter in weight and their forms are altered. Experiments conducted in Poland show that non-typical seed, large, with a high weight per 1000 seeds, and small angular seed carry virus around two times more often than normal seed with normal weight (Ksiazek, 1962). Seed of lupine from Bielanski fodder which was normally formed with a weight per 1000 seeds of 120 grams had a virus infection rate of 4.4%, small angular seed with a weight of 102 grams per 1000 had a rate of 10.3%, and large seed with a weight of 190 grams 8.4% (Blaszczak, 1963). This information is utilizable in practical agriculture in the sense that using appropriate sorting of seed it is possible to a certain degree to limit the extent of virus infection.

Placement of Infected Seed on the Plant

The placement of infected seed on the plant is an interesting problem both from the biological point of view as well as for practical agriculture. If this were completely regular it would be possible to profitably use this knowledge in the field to eliminate sources of disease. As early as 1924 Kendrick and Gardner studied this problem in soybeans infected with mosaic (cited in Couch, 1955). They did not find any correlation between infection of seed and placement on the plant. Likewise Fajardo (1930) did not find any relationship between infection of bean seeds (mosaic virus) and their location on the plant or in individual pods. Couch (1955) asserted that infected lettuce seed (mosaic virus) was not found either on any particular place on the plant nor was there any connection with the time of its formation and disease. Infected seeds at different points varied within the limits of 0 to 35%. Zschau (1962) came to the conclusion that infected yellow lupine seed (narrow-leaf virus) is found both on the main stem and on lateral branches and there is no regularity in the distribution of infected seed in the pods. This question was further studied by Blaszczak (1963). He asserted that among 419 yellow lupine pods examined only 53 pods

contained virus (12.6%). Virus was most often found in four-seed pods and least of all in one-seed pods. This disproves the commonly held opinion among agricultural workers that one-seed pods contain a higher proportion of infected seed. Also there does not seem to be any variation in infection in the pods located on the various whorls of the florescent axis. Infected seed occurs in two- and more seed pods most often as a single seed alongside healthy seeds. At times one finds two or more infected seeds, but there is no regularity in their placement in the pod. Only Harrison (Cited in Gach, 1955) considers that beans in pods which are formed at an early period are more often carriers of the virus than seeds which are formed late. It is necessary to consider in accordance with the statements above that the placement of infected seeds on the plant is irregular and dispersed and therefore there is no possibility of segregating them on this basis from healthy seed destined for later planting.

The Transmission of Viruses by Pollen and in the Ovule

The transmission of virus by seeds assumes that the virus is transmitted to the seeds in the generative cells. Nelson (1933) as the result of crossing two varieties of beans susceptible to typical mosaic virus established that around 1/4 of the ovaries and pollen grains were infected by the virus. The bean variety Robust, highly resistant to typical mosaic was fertilized by pollen from a diseased bean plant (a susceptible variety) and produced in the next generation around 1/4 diseased individual plants. Medina (1961) also asserted that pollen transmitted the typical bean mosaic (and the virus NY 15). Two susceptible bean varieties when self-fertilized produced 42-45% infected seed, while healthy plants fertilized by pollen from diseased plants 86% infected seeds. A bean variety with dominant resistance to typical mosaic, fertilized by diseased pollen produced only healthy seed, while in the case of fertilization by diseased pollen of a variety with resistance determined by recessive genes in F_1 part of the individual plants were diseased. Gilmer and Way (1960) established that there is transmission of viruses in the pollen of fruit trees. Necrotic ringspot virus and plum dwarfing virus were transmitted by pollen at about a rate of 25%. The parental tree (Montmorency) was infected by both viruses and the transmission by pollen of the individual viruses was more frequent than both together. Das and Millrath (1961) proved indisputably than the ringspot virus of drupes is transmitted by pollen from the pumpkin.

Bennett (1959) conducted a number of experiments on the transmission of ringspot virus of lichnis by pollen and seed. He showed that the virus is transmitted in pollen at a rate of 18.6%. This many seeds which developed from healthy plants fertilized by diseased pollen produced diseased seedlings. On the other hand the transmission of the virus was greater (30.7%) in the cases where virus affected plants were fertilized by pollen from healthy plants. This time therefore the virus reached the seed through the ovules. He also fertilized flowers on healthy plants by pollen from virus affected plants and ascertained the percent of infected seed. After collecting the

seed he[illegible]t off the [illegible]lants. The percent of diseased seed obtained by this method varied from 5.1% to 44.6%. However, all the young shoots which sprouted after the trimming of the tree and after the collection of the seeds were healthy. This means that the virus transmitted by the pollen caused a virus infection of parts of the seed, but did not succeed in penetrating to the roots. One must assume that the virus transmitted by the pollen is located in the zygote and is not subject to eventual relocation into the remaining parts of the plant. Crowley (1957) concludes therefore, that the viruses transmitted through the seed can infect neither the macrospore nor the microspore nor the germ.

The Dispersal of Viruses in the Constituent Parts of the Seed

Many virologists have studied the interesting problem of the localization of the virus in the seed. Athow and Bancroft (1959) detected the presence of the tobacco ringspot virus in the cotyledon and the germ of mature soybeans. Viruses were not found in the seed coat. Medina and Grogan (1961) detected the presence of the typical mosaic virus in the germ with the cotyledons of mature beans. Further investigations of this problem were conducted by Quantz (1962) on beans. He dissected mature beans from diseased plants after swelling into the seed coat, cotyledons, and the rest of the germ. The virus of the typical bean mosaic was identified in 93.8% of the germs and in 12.5% of the seed coats. When the cotyledons were examined separately there was a positive isolation of the virus in both the cotyledons and the germ of 54.1%, but from the cotyledons themselves of only 18.9%. In all the presence of the virus was established in 82.3% of the cotyledons and in 75.7% of the germs (excluding the cotyledons). Crowley (1957) obtained similar results. He found the virus of the typical mosaic in 83% of the germs and 21% of the seed coat in beans. The virus of the yellow mosaic, on the other hand, he found in only 7% of the seed coat of beans. He also shows that the cucumber mosaic virus was found in 91% of the seed coats, 49% of the perisperm and 8% of the endosperm in the wild cucumber (Micrampelis lobata) but in only 3% of the seed coats of the cultivated cucumber. As is well known the virus of the yellow mosaic is not transmitted through the seed of the bean, and the mosaic virus of the cucumber is not transmitted through the seed of this species. Blaszczak, investigating narrow-leaf of the yellow lupine (1963), has shown that immature seed from diseased plants are almost all carriers of the virus (around 95%) and the virus occurs then in all parts of the seed but most frequently of all in the seed coat and somewhat less frequently in the cotyledons and in the germ.

Many scientists have studied the transmission of the tobacco mosaic virus in tomato seed. Taylor and his co-workers (1961) established recently in agreement with the results of other experiments (Crowley, 1957) that the tobacco mosaic virus occurs mainly on and in the seed coat, rarely in the endosperm. On the other hand they were not able to isolate the virus from

the germ from either mature or immature seed. Seeds of the tomato are therefore, so to speak, "contaminated" by the virus and when they germinate they cause infection of the young seedlings. In general one can say that the viruses transmitted by seed are localized for the most part in the germ and to a lesser degree in the seed coat. This is compatible with the assertion of Crowley (1957) that only those viruses are transmitted by seed which have the ability to infect the germ.

Disappearance and Survival of Viruses in Mature Seed

Much work has been done to illuminate the curious fact of the "disappearance" of viruses in seeds during the period of their maturation. Among others Stelzner (1942) showed the potato virus X and Y located in seed are subject to inactivation during the period of their maturation, dormancy, and germination. Kausche (1940) found a substance in germinating tobacco seed which inactivates mosaic virus. Zaumeyer and Harter (1943) stated that the southern bean mosaic virus which is not transmitted through seed occurs in relatively high concentrations in the seed of plants which have been systematically infected in the earlier phases of their development, but during the period of maturation and dormancy of the seed they are subject probably to inactivation. This question was examined by Cheo (1955). He confirmed the presence of the virus both in the seed coat and in the germ while as the seed matures up to the 43rd day after florescence the concentration of the virus in the germ significantly increases but it decreases on the other hand in the seed coat. The author suggests that in the developing germ there is an increase in the virus but there is also an accumulation of virus substances from other parts of the plant in the seed. However, four days later when there had been heavy dehydration in the seed one finds only traces of the virus in the seed and it is completely absent in two more days. In completely mature seeds one does not find colored or dry viruses in the cotyledons or in the germ. Both in immature and mature seeds of the bean he found the presence of the substance which reduced the very high infection of the southern mosaic virus. Extracts from mature and germinating bean seeds lowered the infection of the virus 99.7%, that is, almost completely smothered it. Cheo came to the conclusion that the presence of the inhibitor in seeds to a certain degree explains the fact of the disappearance of the virus in the germ, but he concedes the possibility that a number of other elements connected with the physiology of the maturation of the seed may have had a hand in this process.

The disappearance of the cucumber mosaic virus in the seeds of Echninocytus lobata was noted by Crowley (1957). In immature seeds he found 91% virus infection of the seed coat, 49% of the episperm and 8% of the endosperm. There was no virus in the germ. On the other hand after the maturation of the seed the rate of virus infection of the seed coat was reduced to 27% and he was not able to detect the presence of the virus in the episperm and he found it only in 0.7% of the germs. Blaszczak showed that around 95% of the immature seeds ofthe yellow lupine from plants infected with narrow-leaf were carriers of the virus (1963). The virus appeared in

all the seed coats (100%), in 52% of the cotyledons, and 38% of the germs with the greatest concentration of the virus found in the seed coat and a decided decrease in the cotyledons and in the germ. At the same time it is known that narrow-leaf of yellow lupine is transmitted by seed at a rate of a few or at the most of a dozen or so percentage points, which means that during the period of the maturation of the seed there is a great decrease in the number of virus infected seeds. Why it happens that more than 90% of the seed lose their virus and a few percent retain it and pass it on to the young plants which sprout from them is not known. Bojnansky (1963) writes that many inactivating substances appear in the maturing seeds and the virus is subject to hydrolysis and denaturation as the result of the action of the proteases produced by the germ. The Germans suggest (Klinkowski, 1958) that the disappearance of the virus in maturing seeds is connected with the alteration of active albumin into inert albumin. However, neither this hypothesis or any one based on some other substance -- inhibitors active in seeds -- do not adequately explain the phenomenon of the disappearance of viruses in seed during the period of maturation and yet the retention of virus in part of the seeds.

In turn the question arises, how does the virus maintain itself in mature seeds? Because the virus infection of seeds is reduced markedly in the period of maturation, one must conclude that in the period of dormancy this process is continued further. However, it seems that this is not so. Middleton (1944) made a series of plantings of squash seeds and showed that their rate of virus infection held at the same level for three years. Henderson (1931) stated that the tobacco ringspot virus maintained itself in petunia seed at the same level for a series of months and Athow and Bancroft (1959) found the same level of virus infection in soybean by the same virus at the time of seed collection and after nine months of dormancy. The goosefoot mosaic virus was preserved in the seed of Chenopodium murale for a period of 6½ years. No decrease has been found in the percent of virus infected seed in the yellow lupine during the period of dormancy. The presence of the virus has been established in seeds after 5½ years of dormancy. (Blaszczak, 1963). The virus of the western bean mosaic has maintained itself for a three year period in seed (Skotland and Birke, 1961). On the other hand in studies of the viability of bean seed stored for more than 30 years, two seeds germinated and one of them produced a seedling with evidence of mosaic (according to Zaumeyer and Thomas, 1957). This means that viruses established in mature seeds are very persistant and probably maintain themselves in the seeds as long as the seeds preserve their ability to germinate. From the biological point of view the transmission of viruses by seeds is one of the most positive means of guaranteeing their continuity of existance.

Why is the transmission of viruses by seeds limited? There have been a number of hypotheses to explain the process of transmission of viruses by seeds or the converse -- the relatively infrequent occurrence of this phenomenon in nature. Allart (cited in Crowley, 1957) as early as 1915 made the suggestion that virus infection can cause such severe changes in flowers

as to lead to their infertility. This hypothesis can explain the absence of transmission of viruses which cause infertility, such as, for example, the tomato aspermatic virus. This does not explain however why the transmission of viruses is a rarity among plants which in spite of virus infection produce a great number of seeds. Bennett (1936) in turn made the suggestion that the viruses which for the most part cause vascular diseases are not transmitted by seed because between the mother plant and the developing germ there is no vascular linkage. This protects the young germ, according to his explanation, from virus infection and at the same time prevents transmission of those viruses by this method. This hypothesis may be useful in respect to viruses which for the most part cause vascular diseases. On the other hand Duggar (1930) made another hypothesis. He suggested that the transmission of very poisonous viruses in the seeds and also their distribution in the plant is prevented by the inactivating action of a "specific albumin or other specific substances" which is found in the seed. Kausche (1940) suggested that these substances appear during the germination of the seed and then inactivate the viruses (for example, the tobacco mosaic virus). Crowley showed the presence of inhibitors in the seeds of several species of plants (1955) and even in the developing germ (1957) but he did not find any inactivators of virus and therefore he rejected this hypothesis. However, in Europe this opinion is still held (Klinskowski, 1958; Bojnansky, 1963).

Bennett (1936) worked out another theory according to which the non-transmission of viruses by seed may be conditioned by the lack of plasmodesmatic links between the germ and the parent plant. This theory has two assumptions: 1) that the maternal cells-- the microspores and the cells of the germinal sac "flee" from virus infection but that they are also not capable of maintaining the reproduction process of the virus 2) that the one path of intercellular movement of viruses is the plasmodesms.

Crowley (1957) showed that four viruses which are not transmitted by seed are not able to infect the germs of their plant hosts and therefore he accepts the theory of Bennett as most adequately explaining the rarity of transmission of viruses by seed as the result of a lack of plasmodesmatic links between the growing germ and the surrounding tissue. This theory connects the lack of transmission of viruses by seed with the question of the resistance of plants to viruses and the genetic conditioning of the resistance. Crowley believes that if the transmission of a virus by seed is conditioned by its ability for survival in the haploidal generative cells it would be necessary to expect that the genotype both of the host plant as well as the virus would have great significance for this phenomenon. And such indeed is the fact. Couch (1955), as we remember, pointed out that the transmission of the lettuce mosaic by seed is conditioned by the genotype of the plant and of the two known types of the southern bean mosaic virus only one is transmitted by seed (Klinkowski, 1957).

Crowley (1957) supported by the results of the experiments he conducted came to the conclusion that the rarity of the transmission of viruses by seed is not conditioned by any factor. According to his explanation viruses are not transmitted by seeds which cause the death of infected plants, viruses which render the plant incapable of florescence, viruses with limited distribution within the plant-host, and finally those which cannot survive changes occurring in maturing and drying seeds.

Obviously there have been attempts to explain the phenomena of virus transmission by seed. However we are still far from understanding many real questions connected with this problem. Why after maturity does the number of virus infected seeds decrease, for example, in yellow lupine to a few percent but before maturity all the seeds are carriers of the virus? Why in some seeds does the virus "perish" but in others survive in spite of the fact that they were all formed on the same plant? Why do some viruses transmit themselves through the seeds of only some species of plants? The answers to these and many other questions connected with the transmission of viruses by seed could have wide application both in plant culture and in seed science -- in the search for healthy, higher quality seed.

Conclusions

1. The transmission of viruses by seed although limited is found in plant species belonging to various families — for example to Leguminoseae, Solanaceae, Compositae, Rosaceae, Cucurbitaceae, Gramineae and others.

2. The rate of transmission of viruses by seed varies usually within a few percent, although at times the quantity of infected seed is greater and reaches even to several score percent, for example in the typical bean mosaic virus.

3. The percent of virus infected seed on individual plants is very uneven and it is not impossible that this is conditioned by the genotype of the plant.

4. The time of infection of the plant has a strong effect on the virus infestation of seeds. In general one can accept the principle that the earlier a plant is subject to infection the greater will be the percent of seed subject to virus infection.

5. An unfavorable weather period which delays the growth and development of plants also increases the amount of virus infection in seeds.

6. Viruses which are transmitted by seed infect the generative cells and some of them are transmitted by pollen to healthy plants.

7. Viruses transmitted by seed are located and maintain themselves mostly in the germ and less frequently in the seed coat.

8. Immature seeds on infected plants are usually 100% infected. During the period of their maturation the virus "perishes" and the rate of infected seed decreases to a few percent of the total.

9. The placement of virus infected seed on the plant is scattered and shows no regularity.

10. In some cases virus infected seed is poorly formed and shows other morphological changes (e.g., in yellow lupine) which makes it possible to eliminate it from seed materials by sorting.

11. In mature seed viruses are very durable and survive for many years.

Literature

1. Athow Kirk L. and J. B. Bancroft. 1959: "Development and transmission of Tobacco Ringspot Virus in Soybean." Phytopathology, 49: 697-701

2. Bennett C. W. and K. Esau. 1936: "Further studies on the relation of the curly top virus to plant tissues." J. Agr. Res. 53: 595-620

3. Bennett C. W. 1944: "Latent virus of dodder and its effect on sugar beet and other plants." Phytopathology, 34: 77-91

4. Bennett C. W. 1959 "Lychnis Ringspot." Phytopathology 49: 706-713

5. Bennett C. W. and A. S. Costa. 1961. "Sowbane mosaic caused by a seed-transmitted virus." Phytopathology 61: 546-550

6. Bojnansky V. 1963: Virusowe choroby rastlin. (Virus plant diseases) Bratislava

7. Blaszczak W. 1963: "Experiments on narrow-leaf of yellow lupine under Western Polish conditions" Roczniki WSR (Wyzsza Szkola Rolnicza) Poznan, 15: 3-78

8. Cation, Donald. 1959; "Transmission of cherry yellows virus complex through seeds. Phytopathology, 39: 37-40

9. Bretz T. W. 1950: "Seed transmission of the elm mosaic virus." Phytopathology. 40: 3-4

10. Cation, Donald. 1952. "Further studies on transmission of ringspot and cherry yellows viruses through seeds." Phytopathology, 42: 4 (abstr.)

11. Cheo P. C. 1955: "Effect of seed maturation on inhibition of Southern bean mosaic virus in bean." Phytopathology 45: 17-21

12. Cochran L. C. 1950: "Passage of the ring-spot virus through peach seeds." Phytopathology 40: 964 (abstr.)

13. Corbett, M. K. 1958: "A virus disease of lupines caused by Bean Yellow Mosaic Virus." Phytopathology, 48: 86-91

14. Couch H. B. 1955: "Studies on seed transmission of lettuce mosaic virus." Phytopathology, 45: 63-70

15. Crowley N. C. 1955: "The effect of seed extracts on the infectivity of plant viruses and its bearing on seed transmission" Austr. J. Biol. Sci. 8: 56-67

16. Crowley N. C. 1957: "The effect of developing embryos on plant viruses." Austr. J. Biol. Sci. 10: 443-448

17. Crowley N. C. 1957: "Studies on the seed transmission of plant virus diseases" Austr. J. Biol. 10: 449-464

18. Das E. R. and J. A. Milbrath. 1961: "Plant-to-plant transfer of Stone Fruit Ringspot Virus in Squash by Pollination." Phytopathology, 51: 489-490

19. Doolittle S. P. and W. W. Gilbert. 1919: "Seed transmission of cucurbit mosaic by the wild cucumber." Phytopathology, 9: 326-327

20. Duggar B. M. 1930: "The problem of seed transmission of the typical mosaic of tobacco." Phytopathology, 9: 326-327

21. Fajardo T. G. 1930: "Studies on the mosaic of the bean (Phaseolus vulgaris L.)" Phytopathology, 20: 469-494

22. Gilmer R. M. and R. D. Way, 1960: "Pollen transmission of Necrotic Ringspot and Prune Dwarf Viruses in Sour Cherry" Phytopathology, 50: 624-625

23. Grogan R. G. and Roy Bardin. 1950: "Some aspects concerning seed transmission of lettuce mosaic virus" Phytopathology 40: 965 (Abstr.)

24. Henderson R. G. 1931: "Transmission of Tobacco Ring Spot by seed of Petunia." Phytopathology, 21: 225-229

25. Kausche G. K. 1940 "Concerning an inactivating substance of virus protein in the seed of Nicotiana tabacum var. Samsun" (In German) Biol. Zbl. 60: 423-438

26. Kendrick J. B. 1934: "Cucurbit mosaic transmitted by muskmelon seed." Phytopathology. 24: 820-823

27. Klinkowski M. 1958. Pflanzliche Virologie (Plant Virology) Band I, II Berlin

28. Ksiazek, D. 1962 "Studies on the virus diseases of lupines -- narrow-leaf, brown-disease, and mosaic" Acta Agrobot. 12: 287-322

29. Mastenbroek C. 1942: "Enkele veldwaarnemingen over virus-ziekten van Lupine en enn onderzoek over haar mozaik-ziekten" T. Plantenziekt. 48. According to Rev. Appl. Myc. 25: 68 (1946)

30. McKinney H. H. 1951: "A seed-borne virus causing false stripe in barley. Phytopathology 41: 563-564 (Abstr.)

31. McLean D. M. 1941: "Studies on mosaic of cowpeas, Vigna sinensis." Phytopathology 31: 420-430

32. Medina Crispin A. and R. G. Grogan. 1961: "Seed transmission of bean mosaic viruses" Phytopathology 51: 452-456

33. Merkel L. 1929: "Contributions to the study of mosaic disease in the papilionaceous family" (In German) Z. Pflanzenkrankh. und Pflanzenschutz 39, 289-347

34. Middleton J. T. 1944: "Seed transmission of squash mosaic virus." Phytopathology 34: 405-410

35. Nelson Ray and E. Down. 1933: "Influence of pollen and ovule infection in seed transmission of bean mosaic" Phytopathology, 23: 25

36. Pozdena J., Snobodova J., Petru E., Limberk J. and C. Blattny. 1955: "A Contribution to the Study of the Pea Mosaic in the Czechoslovak Socialist Republic." Fol. Biol. 1: 298-309

37. Quantz L. 1962: "Towards the determination of the typical bean mosaic in bean seed with the aid of a scale test." Nachrichtenbl. Dtsch. Pflanzenschutzd. (Braunschweig) 14: 49-54

38. Reddick D. and V. B. Steward. 1919: "Transmission of the virus of bean mosaic in seed and observation on thermal deathpoint of seed and virus." Phytopathology 9: 445-450

39. Sing G. P., Arny D. C. and G. S. Pound. 1960: "Studies on the stripe mosaic of barley, including effects of temperature and age of host on disease development and seed infection." Phytopathology 50: 290-296

40. Skotland C. B. and D. W. Burke. 1961: "A Seed-borne bean virus of wide host range." Phytopathology, 51: 565-568

41. Smith K. M. 1957: A Textbook of plant virus diseases. Boston

42. Stelzner G. 1942: "Concerning the question of the dissemination of virus through seed, especially of X- and Y- and leaf curl virus of potato." Züchter 14: 225-234

43. Taylor R. H., Grogan R. G., and K. A. Kimble. 1961: "Transmission of Tobacco Mosaic Virus in Tomato Seed" Phytopathology, 51: 837-842

44. Zaumeyer W. J. and L. L. Harter. 1943: "Two new virus diseases of beans" J. Agric. Res., 67: 305-328

45. Zumeyer W. J. and H. Rex Thomas. 1957: A Monographic study of Bean diseases and methods for their control. Techn. Bull. No 868. USDA

46. Zawadzki S. and J. Grzybczak. 1962 "Experiments on mosaic disease of yellow lupine" Hodowla Roslin, Alim., i Nasiennictwo. (Plant Culture, Acclimatization, and Seed) 6: 227-250

47. Zschau K. 1960. "Concerning the transmission of the cucumber mosaic virus by the seeds of Lupinus luteus." Tagungsberichte nr 29, Wissenschaftliche Tagung am 16. Dezember 1959 in Kleinmachnow. Berlin 41-51

48. Zschau K. 1962: "Experiments and observations on the transmission by seed of the mosaic diseases of Lupines, in particular the yellow lupine" Nachrichtenbl. Dtsch. Pflanzenschutzd. (Berlin), 16: 1-7

- END -